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Modulation of Atrioventricular Conduction by Ablation of the "Slow" Atrioventricular Node Pathway in Patients With Drug-Refractory Atrial Fibrillation or Flutter

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Objectives. **We hypothesized that modulation of atrioventricular (AV) node conduction, allowing a reduction in ventricular rate during atrial fibrillation or flutter without affecting AV conduction during sinus rhythm, might be achieved through ablation of the "slow" AV node pathway.**

Background. **In patients with atrial fibrillation or flutter not amenable to a direct atrial approach, ablation of the His bundle is performed to induce complete AV block. This procedure causes pacemaker dependence.**

Methods. **Fourteen patients with drug-refractory paroxysmal atrial flutter or fibrillation underwent ablation of the slow AV node pathway. Radiofrequency current was delivered in six patients during sinus rhythm, in six during atrial flutter and in two during atrial fibrillation.**

Results. **The anterograde effective refractory period of the AV node was prolonged from 270 ± 50 (mean ± SD) to 390 ± 87 ms ($p = 0.005$) and the Wenckebach cycle from 346 ± 33 to 458 ± 75 ms ($p = 0.004$) in six patients during sinus rhythm. Mean AV ratio increased from 1.6 ± 0.5 to 3.0 ± 0.6 ($p = 0.02$) in six patients with atrial flutter. Mean ventricular rate decreased from 157 ± 38 to 67 ± 10 beats/min in two patients with atrial fibrillation. Complete AV block was induced in two patients (transient in one, permanent in one). During a follow-up period of 5.8 ± 3.5 months, 11 patients experienced a recurrence of atrial fibrillation at 60 to 95 beats/min. No patient had progression to any degree of AV block.**

Conclusions. **Ablation of the slow AV node pathway allows reduction of ventricular rate during atrial fibrillation or flutter while maintaining intact AV conduction during sinus rhythm. Modulation of AV node conduction is effective in most patients as an alternative to His bundle ablation for control of ventricular rate in paroxysmal atrial fibrillation or flutter.**

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Patients with drug-refractory atrial fibrillation or flutter are currently considered candidates for transvenous ablation of the His bundle for the purpose of inducing complete atrioventricular (AV) block (1–3). However, the resulting pacemaker dependence represents the major drawback of the procedure, particularly in patients with paroxysmal arrhythmias in whom a physiologic AV activation sequence would be desirable during sinus rhythm.

Direct atrial ablation allows successful termination of typical atrial flutter with a low incidence of short-term recurrence (4–6). However, most patients with atrial flutter have structural heart disease, which, in association with atypical atrial flutter or atrial fibrillation, is less likely to lead to successful long-term results because of extensive pathologic changes in the atria (7,8).

In this setting a modification of AV node conduction properties, aimed at a reduction of ventricular rate during atrial fibrillation or flutter, could be beneficial. Furthermore, to be clinically useful the procedure should allow regular AV conduction during sinus rhythm. Electrophysiologic studies (9) of AV node conduction after selective radiofrequency catheter ablation of either the "fast" or the "slow" AV node pathway have shown a prolonged anterograde effective refractory period and Wenckebach cycle after ablation of the slow pathway, without changes in the AH interval during sinus rhythm. By contrast, although ablation of the fast pathway prolongs the AH interval, it does not change the Wenckebach cycle and the refractory period of the AV node (9).

These data support the hypothesis that the "slow" pathway plays an important role in AV conduction at fast rates and constitute the rationale for it as a target for ablation to achieve modulation of AV node conduction.

This study evaluates the immediate results and short-term follow-up data of slow AV node pathway ablation to control ventricular rate in patients with drug-refractory paroxysmal atrial fibrillation or flutter.

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Table 1. Clinical Characteristics of the 14 Study Patients

Pt No./Gender	Age (yr)	Underlying Disease	Presenting Atrial Arrhythmia	Max HR (beats/min)	Symptoms
1/M	60	CAD	Atrial flutter + fibrillation	145	Angina
2/F	69	Mitral prothesis	Atrial tachycardia + fibrillation	230	Acute pulmonary edema
3/M	63	Hypertension	Atrial flutter + fibrillation	160	Palpitations
4/F	45	Mitral prothesis	Atrial flutter + fibrillation	250	Syncope
5/M	65	Hypertension	Atrial flutter + fibrillation	170	Acute pulmonary edema
6/M	36	None	Atrial flutter + fibrillation	140	Palpitations
7/M	48	None	Atrial flutter + fibrillation	135	Palpitations
8/M	40	None	Atrial fibrillation	185	Palpitations
9/M	52	DCM	Atrial flutter*	150	Acute pulmonary edema
10/F	40	HPCM	Atrial flutter + fibrillation	150	Syncope
11/F	69	Hypertension	Atrial tachycardia + fibrillation	190	Angina, dizziness
12/M	57	Mitroaortic disease	Atrial fibrillation	145	Acute pulmonary edema
13/F	60	Operation for ASD	Atrial flutter + fibrillation	140	Palpitations
14/F	68	Hypertension	Atrial fibrillation	160	Palpitations, dizziness

*Two types of flutter. ASD = atrial septal defect; CAD = coronary artery disease; DCM = dilated cardiomyopathy; F = female; HPCM = hypertrophic cardiomyopathy; M = male; Max HR = maximal heart rate; Pt = patient.

Methods

Patients. The study included 14 patients (8 men, 6 women; 36 to 69 years old, mean [±SD] age 55.1 ± 11.5) with recurrent drug-refractory paroxysmal atrial tachycardia, flutter or fibrillation, leading to severe symptoms (syncope in two; pulmonary edema in four; angina, dizziness and palpitations in eight). In all patients multiple types of arrhythmias (e.g., different configurations of atrial flutter or fibrillation and flutter) were documented for each patient at different times. For this reason the patients included in the study were not considered candidates for direct atrial ablation. Mean left atrial size was 49 ± 8 mm (range 36 to 65); mean left ventricular ejection fraction was 47 ± 9% (range 20% to 55%). The clinical characteristics of the patients are summarized in Table 1; details of therapy are presented in Table 2.

Electrophysiologic procedures. All patients were sedated by an intravenous bolus of morphine and diazepam; invasive arterial blood pressure and oxygen saturation were continuously monitored throughout the procedure. Multipolar electrode catheters were positioned in the right atrium, right ventricle and coronary sinus and across the tricuspid ring to record the His bundle electrogram. An additional 4-mm tip-deflectable catheter was placed over the tricuspid ring to perform mapping of the right atrial posteroseptal region and for later ablation. Multiple surface electrocardiographic (ECG) leads and intracavitary tracings were simultaneously recorded on a Siemens T 16 polygraph at a paper speed of 200 mm/s; electrical stimulation was provided by a Medtronic 5320 Programmable Stimulator.

The following variables were analyzed at baseline and after ablation: anterograde effective refractory period of the AV node and the Wenckebach cycle (in six patients in sinus rhythm); AV conduction ratio and maximal heart rate during atrial flutter or tachycardia (six patients); and mean ventricular rate over a 30-min period in the two patients with atrial fibrillation. Furthermore, in six patients in whom ablation was performed during sinus rhythm, the AV conduction ratio or maximal heart rate was evaluated during atrial flutter or tachycardia or during atrial fibrillation induced after the procedure.

Ablation procedure. Ablation performed during sinus rhythm in six patients, atrial flutter or tachycardia in six and atrial fibrillation in the remaining two. Radiofrequency current was delivered in the unipolar mode between the ablation catheter tip and an electrodispersive skin patch placed posteriorly, using a radiofrequency generator providing 550-kHz unmodulated energy (American Cardiac Ablation Company, model Liz 88). The applied voltage ranged from 40 to 60 V; impedance was continuously monitored by meter, and mean values ranged from 90 to 110 ohms; the resulting power was 15 to 30 W; pulses duration was 15 to 60 s. Energy delivery was discontinued if an impedance increase >120 ohms was observed.

The target for ablation of the slow AV node pathway during sinus rhythm was a high frequency potential, closely following low amplitude atrial activation, preceding the His bundle deflection by 35 ± 12 ms (range 20 to 50) (Fig. 1), occurring synchronously or later than atrial activity recorded in the proximal coronary sinus, as previously described by Jackman et al. (10) for ablation of AV junctional tachycardia. Mean slow pathway potential ventricular electrogram ratio was 0.75:1 (range 0.6:1 to 1:1). This potential was usually recorded in the low posterior septum (Fig. 2), slightly anterior to the coronary sinus os. Induction of a junctional rhythm with 1:1 ventriculoatrial (VA) conduction was considered a criterion of efficacy (10,11). Radiofrequency energy was interrupted if no junctional rhythm ensued within 10 s. When junctional rhythm occurred, energy was delivered for 30 to 45 s, until disappearance of the junctional rhythm. The catheter was then moved, in the same region, to record a similar potential, and energy was again delivered until the appearance of a junctional rhythm could be documented. The procedure was repeated several

Table 2. Therapy, Maximal Documented Heart Rate and Related Symptoms Before and After Ablation

	Before Ablation			After Ablation		
Pt No.	Therapy	Max HR (beats/min)	Symptoms	Therapy	Max HR (beats/min)	Symptoms
1	Quinidine, disopyramide, flecainide, amiodarone, verapamil, digoxin	145	Angina	Flecainide (50 mg bid)	90	—
2	Flecainide, metoprolol, verapamil, digoxin	230	Acute pulmonary edema	Digoxin (0.125 mg die)	76	—
3	Flecainide, metoprolol	160	Palpitations	Captopril (50 mg die)	—	—
4	Propafenone, flecainide, metoprolol, sotalol, amiodarone	250	Syncope	Sotalol (60 mg bid)	85	Palpitations
5	Quinidine, disopyramide, flecainide, verapamil, digoxin	170	Acute pulmonary edema	Captopril (50 mg die)	70	Palpitations
6	Propafenone, flecainide, sotalol, amiodarone, verapamil	140	Palpitations	—	—	—
7	Disopyramide, propafenone, flecainide, amiodarone, verapamil	135	Palpitations	—	90	—
8	Metoprolol, sotalol, amiodarone, verapamil	185	Palpitations	Nadolol (20 mg bid)	75	—
9	Flecainide, amiodarone, verapamil, digoxin	150	Acute pulmonary edema	Digoxin (0.25 mg die)	90	—
10	Metoprolol, amiodarone, verapamil	150	Syncope	Flecainide (50 mg bid)	70	Palpitations
11	Quinidine, disopyramide, propafenone, amiodarone, verapamil	190	Angina, dizziness	Enalapril (20 mg die)	70	—
12	Amiodarone, verapamil, digoxin	145	Acute pulmonary edema	Digoxin (0.125 mg die)	60	—
13	Disopyramide, flecainide, verapamil	140	Palpitations	—	—	—
14	Quinidine, disopyramide, propafenone, verapamil	160	Angina, dizziness	—	80	—

bid = twice a day; die = daily; other abbreviations as in Table 1.

times in each patient (range 5 to 15) before undertaking measurement of AV node conduction properties by programmed atrial stimulation or overdrive pacing. Although target values for AV node refractoriness or Wenckebach point were not defined, the effort was made to modify the AV conduction ratio so as to avoid 1:1 conduction at paced rates >120 beats/min.

When the procedure was performed during atrial flutter or tachycardia, mapping of the same area was performed to record an electrogram, characterized by atrial activity including a high frequency spike (Fig. 3). This probably cannot be considered a recording of the slow pathway potential, but it can be used to localize the base of the triangle of Koch. Several radiofrequency pulses were delivered with the goal of obtaining a stable increase in AV conduction ratio.

In the two patients with atrial fibrillation, selection of the ablation site was guided by anatomic markers, with the ablation catheter placed anteriorly to the coronary sinus os, 1 to 1.5 cm below the His bundle, to record the irregular activity of atrial fibrillation with the highest possible amplitude. An initial increase followed by a sustained slowing of the ventricular rate after the onset of radiofrequency delivery (Fig. 4) was considered evidence of ablation of the slow pathway. Several current pulses were delivered at closely surrounding places until a permanent slowing of the heart rate was achieved.

All patients were monitored for 1 h in the electrophysiologic laboratory to ensure stability of the results. Isoproterenol (1 to 3 μg/min) was infused in three patients in whom a stable 4:1 AV conduction pattern was induced after ablation to ensure capability to increase ventricular rate on sympathetic stimulation. Echocardiography was performed immediately after the procedure and on the following day.

Patient follow-up. Patients were discharged from the hospital 4 to 6 days after the procedure; during the hospital stay they were monitored by telemetry. Aspirin (325 mg daily) was prescribed for 2 months; digoxin or beta-adrenergic blocking agents were prescribed when needed in patients who had been taking the same therapy before ablation. Details of therapy are reported in Table 2. After discharge, an outpatient clinic visit was scheduled at 1 month and then at 2-month intervals. A standard ECG and a 24-h Holter ECG were obtained at each visit. Patients were also requested to record an ECG in the event of palpitations.

Statistical methods. Results are expressed as mean value ± SD. A Student *t* test was used to assess the significance of the observed changes in the refractory period and Wenckebach cycle.

Results

Electrophysiologic data. Detailed values of the AV node refractory period, Wenckebach cycle, AV ratio and mean

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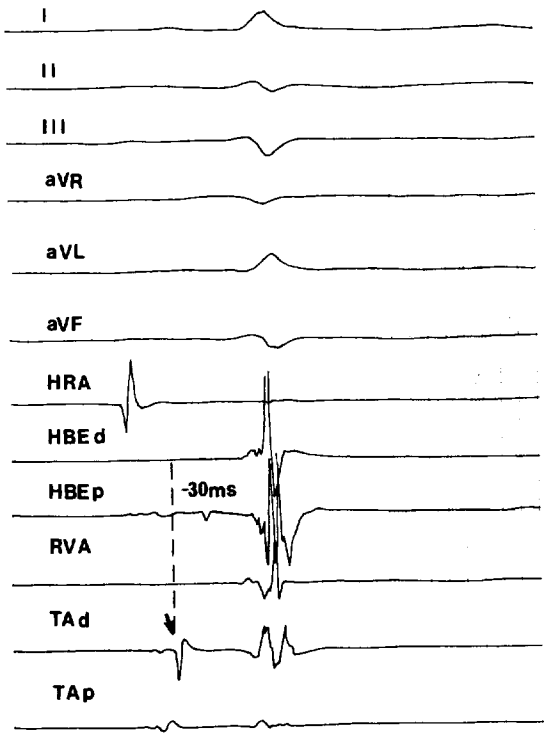


Figure 1. Simultaneous recordings of six standard surface electrocardiographic leads and multiple intracavitary electrograms (Patient 3). **Arrow** points to the potential used to localize the slow atrioventricular node pathway ablation site during sinus rhythm. In the tricuspid annulus recorded through the distal electrode pairs of the catheter (TAd), a sharp spike follows low amplitude atrial activity; the spike occurs 30 ms before the His bundle deflection. Paper speed 200 mm/s. d (p) = distal (proximal) electrode pairs of the catheter; HBE = His bundle electrogram; HRA = high right atrium; RVA = right ventricular apex.

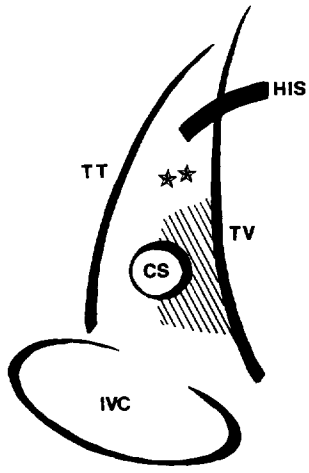
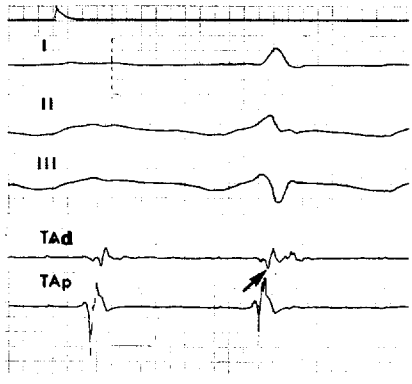


Figure 2. Schematic illustration of the triangle of Koch. **Hatched area** represents the posterior part of the septum, where the slow pathway potential was recorded and radiofrequency energy was delivered to achieve modulation of the atrioventricular (AV) conduction. **Stars** indicate the sites where complete AV block (transient in one patient and permanent in the other) was induced. CS = coronary sinus; His = His bundle; IVC = inferior vena cava; TT = tendon of Todaro; TV = tricuspid valve.

ventricular rate before and after ablation are presented in Table 3. In six patients the procedure was performed during sinus rhythm, thus allowing evaluation of the changes in AV node physiology. The anterograde effective refractory period increased from 270 ± 50 to 390 ± 87 ms ($p = 0.005$) and the Wenckebach cycle from 346 ± 33 to 458 ± 75 ms ($p = 0.004$). An episode of atrial flutter or fibrillation was induced at the end of the procedure in all six patients, documenting a marked decrease in ventricular rate during the arrhythmia. The mean number of radiofrequency pulses was 10.7 ± 4.0 in this group of patients. When radiofrequency was delivered during atrial flutter (Fig. 5) or tachycardia (six patients), the AV conduction ratio increased from 1.6 ± 0.5 to 3.0 ± 0.6 ($p = 0.02$); maximal heart rate decreased from 172 ± 53 to 93 ± 26 beats/min ($p = 0.04$). The mean number of radiofrequency pulses to achieve this result was 8.2 ± 2.6. In one patient (Patient 6) no changes in AV ratio were observed after three radiofrequency pulses delivered to the posteroseptal region. The ablation catheter was moved slightly superiorly, closer to the His bundle recording site. Complete AV block occurred within 4 s of radiofrequency delivery, and despite the recovery of a temporary 4:1 AV conduction pattern, complete AV block ensued 12 h later, and a permanent ventricular rate-responsive (VVIR) pacemaker was implanted. Temporary complete AV block was also induced in a second patient (Patient 14) in whom the procedure was initially performed during sinus rhythm. The delivery of radiofrequency pulses caused an increase in the effective refractory period from 230 to 320 ms and a prolongation of the Wenckebach cycle from 340 to 380 ms. However, the ventricular rate during atrial fibrillation induced afterward was 130 to 140 beats/min. Sinus rhythm could not be recovered after two direct current shocks in this patient. Additional radiofrequency pulses were delivered during atrial fibrillation in an attempt to reduce ventricular response. However, complete AV block resulted after the third radiofrequency pulse, delivered 1.5 cm posterior to the His bundle recording site, with resumption, 2

Figure 3. **Arrow** indicates the electrogram recorded at the ablation site in a patient during atrial flutter (Patient 1). A distinct **spike** follows local atrial activation. Paper speed 200 mm/s. Abbreviations as in Figure 1.



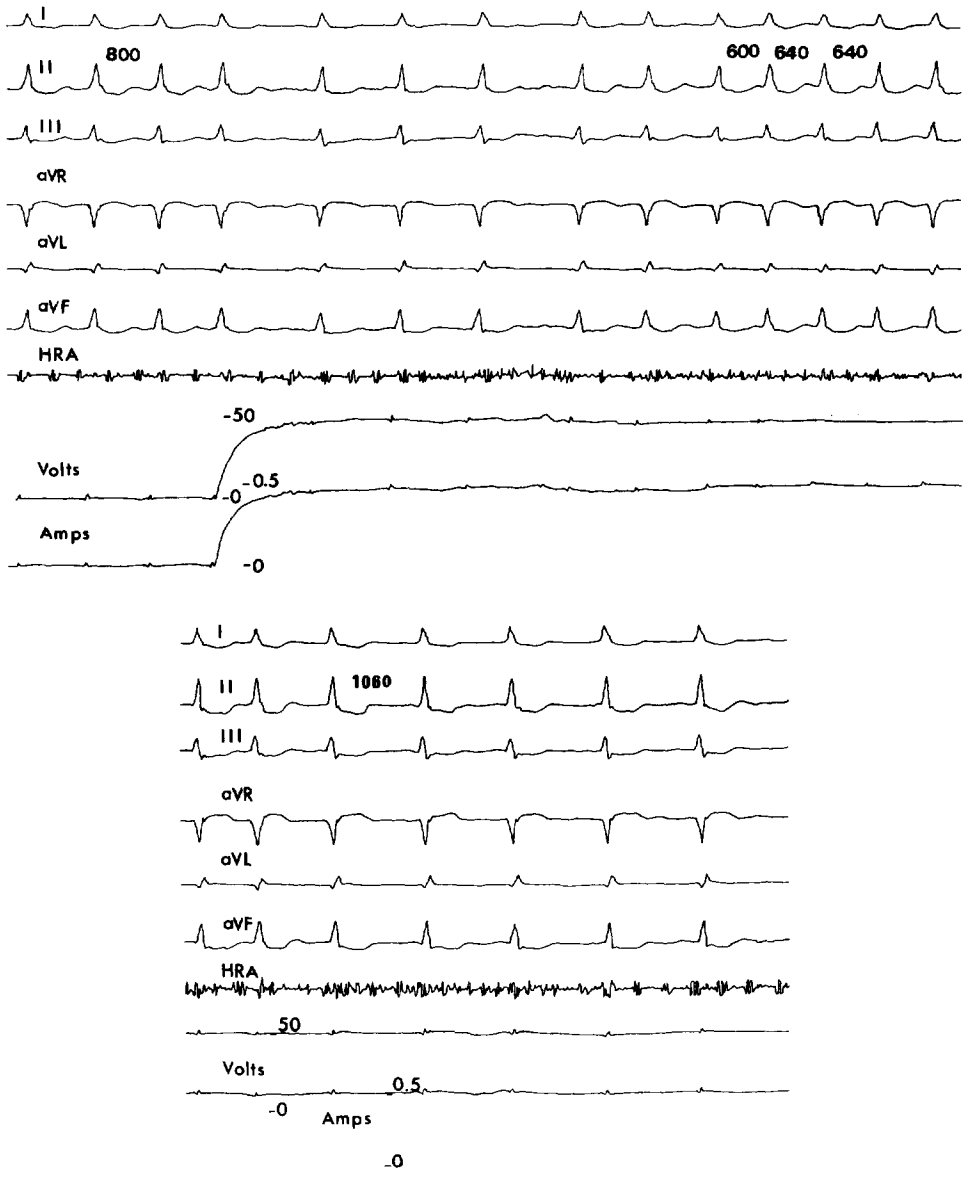


Figure 4. Effects of radiofrequency energy pulse targeted on the "slow" atrioventricular node pathway during atrial fibrillation in Patient 14. The ablation site in this patient was selected by means of anatomic landmarks. **Top,** A marked shortening of the RR cycle is observed 3 s after current delivery. **Bottom,** After 30 s from onset of radiofrequency delivery, mean ventricular cycle is prolonged to 1,060 ms (shown in **lead II**). Mean ventricular rate in the following 30-min period ranged from 60 to 70 beats/min. Paper speed 50 mm/s. HRA = high right atrium.

days later, of sinus rhythm with a 1:1 AV conduction ratio. A VVIR pacemaker was subsequently implanted for safety reasons. In-hospital ECG monitoring revealed constant normal AV conduction (PQ interval 160 ms) and a recurrence of atrial fibrillation with a ventricular rate of 60 to 65 beats/min.

Despite problems with reliable evaluation of ventricular rate in the setting of atrial fibrillation, mean ventricular rate, calculated over a 30-min period, decreased from 157 ± 38 to 67 ± 10 beats/min after 9.5 ± 4.9 radiofrequency pulses in the two patients (Patients 8 and 12) with this arrhythmia. A transient increase in ventricular rate, followed by a late persistent slowing, was considered a marker of successful slow pathway ablation.

At termination of the procedure, sinus rhythm was present in 8 of 14 patients, and the AH interval was normal in four patients (Patients 1, 2, 3 and 5) and slightly prolonged in four (Patients 4, 10, 11 and 13). However, in three of the latter patients (Patients 10, 11 and 13), the AH interval was unaffected by the procedure. In the remaining patient (Patient 4), ablation was performed during atrial flutter, and baseline control AH values are not available; the PR interval during sinus rhythm remained unchanged with respect to preablation tracings. Mean fluoroscopic time was 39 ± 9 min.

In-hospital monitoring. All patients were monitored by telemetry for 4 to 6 days after the procedure. Regular sinus rhythm was documented in all patients without disturbances of AV conduction, with the exception of Patient 6 who had a permanent pacemaker implanted 2 days after ablation. Recurrences of atrial arrhythmia were documented, either by telemetry or by Holter recording, in seven patients (Patients 1, 4, 7, 9, 11, 12 and 14) at a variable rate from 60 to 95 beats/min. The

Table 3. Electrophysiologic Features Before and After Ablation

Pt No.	Presenting Arrhythmia	No. of Pulses	Rx Time (min)	AV Ratio		Max HR* (beats/min)		Wenkebach Cycle (ms)		ERP (ms)		AH Interval (ms)	
				Before	After	Before	After	Before	After	Before	After	Before	After
1	Atrial flutter	6	50	2:1	3–4:1	135	90	—	—	—	—	80	80
2	Atrial tachycardia	8	36	1–2:1	3–4:1	230	76	—	—	—	—	80	80
3	Sinus rhythm	6	33	2:1	4:1	160	80	350	400	220	350	60	60
4	Atrial flutter	10	53	1–2:1	4:1	250	62	—	—	—	—	—	140
5	Sinus rhythm	9	21	2:1	4:1	120	60	300	410	250	340	60	60
6	Atrial flutter	3 + 1	37	2:1	2:1	140	140	—	—	—	—	—	CAVB
7	Atrial flutter	9	51	2:1	3–4:1	130	90	—	—	—	—	—	—
8	Atrial fibrillation	6	30	—	—	185*	75*	—	—	—	—	—	—
9	Atrial flutter	5	50	2:1	3:1	150	100	—	—	—	—	—	—
10	Sinus rhythm	15	37	—	—	150	95	340	460	290	340	130	130
11	Sinus rhythm	13	42	2:1	4:1	190	70	360	550	290	520	110	110
12	Atrial fibrillation	13	39	—	—	130*	60*	—	—	—	—	—	—
13	Sinus rhythm	6	31	—	—	140	80	400	550	350	480	100	100
14	Sinus rhythm + atrial fibrillation	9 +3†	48	—	—	160*	140*	340	380	230	320	80	CAVB‡

*Mean heart rate (HR) over a 30-min period in patients with atrial fibrillation. †Number of pulses during atrial fibrillation. ‡Transient complete atrioventricular (AV) block (CAVB). ERP = effective refractory period; Pt = patient; Rx = treatment.

echocardiogram obtained the day after each procedure remained unchanged, without signs of pericardial effusion.

Follow-up. All patients were followed up at the outpatient clinic at 1 month and then at 2-month intervals after ablation for a mean of 5.8 ± 3.5 months (range 2 to 12). Recurrences, either symptomatic or asymptomatic, of atrial tachycardia, flutter or fibrillation were documented in 11 of 14 patients (Patients 1, 2, 4, 5, 7 to 12 and 14) during the follow-up period. Three patients (Patients 4, 5 and 10) had paroxysms of palpitations, and all three had documented recurrence of atrial fibrillation with a ventricular rate of 75 ± 8 beats/min. These patients had presented with syncope (Patients 4 and 10) or acute pulmonary edema (Patient 5) that did not recur at follow-up. In the remaining eight patients, short-duration asymptomatic atrial tachycardia, atrial flutter or atrial fibrillation with a ventricular rate of 78 ± 10 beats/min was documented during Holter monitoring or scheduled follow-up visits. A follow-up exercise ECG was obtained during atrial fibrillation in two patients (Patients 8 and 12); peak heart rate was 120 and 125 beats/min respectively. In no patient was there progression to high grade AV block.

Discussion

The results of this study support the possibility of modulation of AV conduction by radiofrequency ablation of the "slow pathway." The procedure is feasible in most patients and allows a substantial reduction in ventricular rate during atrial tachycardia, flutter or fibrillation without affecting AV conduction during sinus rhythm.

Modulation versus ablation of AV junction. Radiofrequency ablation of the AV junction for the purpose of inducing a permanent, complete AV block is effective, but results in lifetime pacemaker dependence (1–3). This represents the major limitation of the procedure in patients with paroxysmal arrhythmias and results in loss of the physiologic AV activation sequence during sinus rhythm. The possibility of slowing the ventricular rate during atrial fibrillation without affecting conduction during sinus rhythm—modulation of AV conduction—was initially investigated by Duckeck et al. (12), by delivery of graded radiofrequency energy to the anterior region of the interatrial septum, close to the His bundle recording site. The end point of the procedure was a 50% increase in AH interval or a prolongation of the Wenckebach cycle to 400 ms. The results were disappointing because of a high rate of immediate or late AV block and low clinical efficacy during follow-up. It was thought that modulation of AV conduction could not be achieved in a reproducible fashion and was related to nonspecific damage to the AV node–His bundle conduction system. However, ablation of the slow pathway has more recently been shown to effectively reduce ventricular rate during spontaneous (13,14) or induced (15) atrial fibrillation, without causing complete AV block.

Is there always a "slow AV node pathway"? The results of the present study are not surprising in view of the changes in AV node conduction that occurred after selective radiofrequency catheter ablation of the "slow pathway," as previously described in patients with AV junctional tachycardia (9). In these patients, the presence of a dual AV node physiology is considered to represent the substrate for reentry. However, use of the slow pathway as a target for ablation in patients without dual AV node pathways implies that it is part of the "normal" AV node. Dual AV node physiology can also occur in the absence of AV node reentry tachycardia (16), as initially described by Denes (17). Recent pathologic studies (18) have also shown the absence of differences in AV node structures in patients with or without evidence of dual AV node physiology. The presence of slow pathway potentials was described by

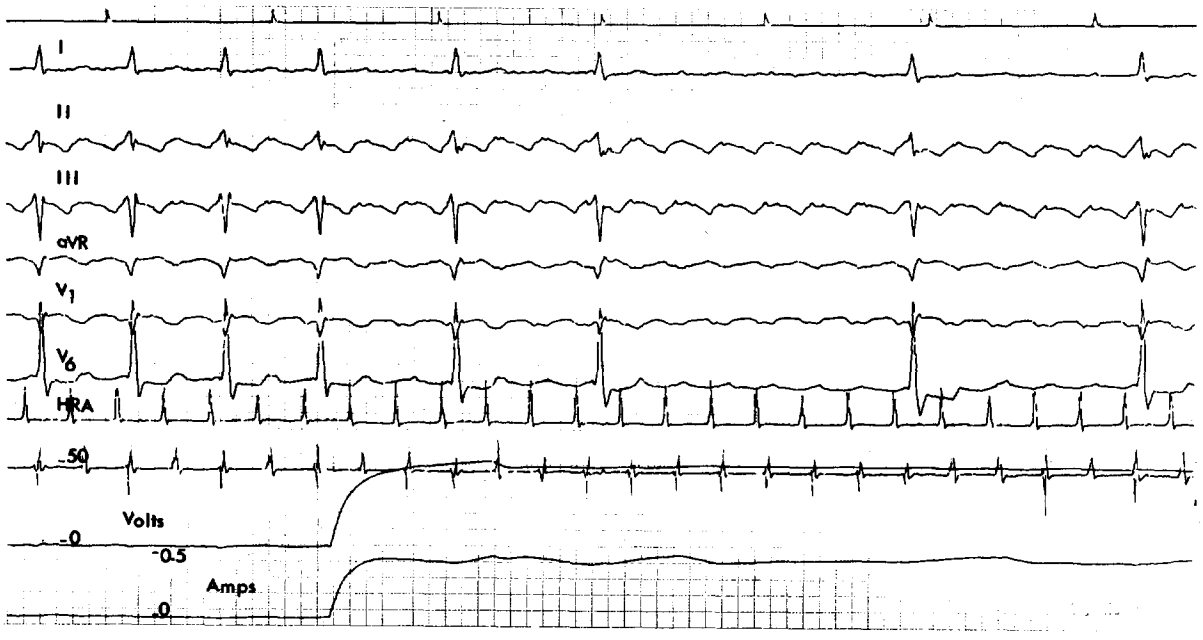


Figure 5. During an episode of typical atrial flutter with 2:1 atrioventricular (AV) conduction (first three cycles), delivery of radiofrequency current to the slow AV node pathway in this patient (Patient 1) causes a sudden increase in AV ratio to 3:1 for two cycles, followed by a 7:1 and a 5:1 cycle; the AV ratio in the following beats remained stable at 4:1. Paper speed 50 mm/s. HRA = high right atrium.

Haissaguerre et al. (19) in 80% of control patients without AV node reentry tachycardia. It is possible that these potentials do not represent activation of a slow AV node pathway; rather these could originate from atrial fibers joining the AV node from beneath the coronary sinus or fossa ovalis. The immediate electrophysiologic effect occurring during radiofrequency delivery in sinus rhythm—induction of an active junctional rhythm with 1:1 VA conduction over the AV node—was in keeping with that commonly observed during slow pathway ablation for AV junctional tachycardia. Similarly, the rapid increase in degree of AV ratio conduction after radiofrequency delivery during atrial flutter or tachycardia confirms that the ablated area plays a key role in allowing AV conduction at high rates. The AH interval remained unaffected by the procedure in all patients in whom it was measured. These observations describe the electrophysiologic effects expected as a result of ablation of the slow pathway. It should be stressed that none of the patients had clinical or electrophysiologic dual AV node physiology. Our data therefore support the concept that the currently considered "slow pathway" is an atrial input with distinct conduction properties. This is probably part of the normal physiology of the AV node and is located in most cases in the posteroinferior atrial septum, anterior to the ostium of the coronary sinus.

End points of the procedure. Prolongation of the Wenckebach cycle to 500 ms was a presumed end point because it was thought that this would allow a marked reduction in ventricular rate should atrial arrhythmia recur. Although the anterograde refractory period of the AV node and Wenckebach point were not systematically assessed after each radiofrequency delivery, it appeared clear that the modification of these conduction variables was a stepwise phenomenon, taking place gradually and requiring several pulses of energy at adjacent sites. Also, the degree of prolongation of the AV node effective refractory period and Wenckebach point required to achieve the modulation is greater than that observed after ablation of the slow pathway for the treatment of AV junctional tachycardia. In the series published by Jazayeri et al. (9), the mean anterograde AV node refractory period increased from 245 to 280 ms and the Wenckebach cycle from 302 to 337 ms after slow pathway ablation. In the present series the values increased from 270 to 390 ms and from 346 to 458 ms, respectively. In all cases the changes in AV conduction documented at the termination of the procedure reliably predicted ventricular rate during atrial fibrillation or flutter, as later confirmed in 11 of our patients during recurrence of spontaneous arrhythmia.

When modulation was attempted during atrial flutter or tachycardia, the increase in AV ratio was a reliable guide for defining the outcome of each energy application. Similar to that observed during sinus rhythm, attainment of a stable increase in AV ratio required multiple pulses.

Therefore, AV node modulation seems to be a quantitative procedure that allows different levels of change in conduction variables to be obtained. The procedure does not appear to involve simple resection of a distinct "slow pathway"; rather, it implies possible extensive damage to the atrial tissue that conveys distinct functional inputs to the AV node, which may account for conduction at rapid rates.

Failures of the procedure. Complete AV block was induced at the termination of the procedure in two patients

because of insufficient reduction in ventricular rate during atrial flutter in one patient and atrial fibrillation in the other. In both patients the AV block resulted from repeated attempts to reduce ventricular rate during flutter or fibrillation after extensive ablation. However, in one of these patients, 1:1 AV conduction ratio resumed 3 days later and remained stable during the follow-up period (2 months). From a physiologic point of view, these data mean that in at least 15% of the candidates for AV node modulation, ablation of the slow pathway or "posteroinferior right atrial input" is ineffective. It is possible that, in these patients, other inputs, from the anterior septum or from the left side, may sustain conduction to the AV node at high rate; their electrophysiologic characterization may be helpful for achieving modulation in patients in whom the approach described herein failed.

Results were similar when ablation was performed during sinus rhythm, atrial flutter or fibrillation. However, the higher incidence of complete AV block that occurred during atrial fibrillation or flutter suggests that the procedure is better done in sinus rhythm, when a precise recording of the potential for the ablation site is possible. When ablation is performed during atrial flutter or atrial fibrillation, the location of the ablation target is guided by "anatomic" landmarks only and is less precise. Furthermore, monitoring of junctional rhythm and retrograde VA conduction during energy delivery is helpful in avoiding permanent AV block and can be recognized clearly only during sinus rhythm (10,11).

Conclusions. Modulation of AV node conduction can be proposed as an alternative to His bundle ablation for control of drug-refractory paroxysmal atrial fibrillation or flutter not amenable to direct atrial ablation. Furthermore, it can be performed as in addition to atrial ablation for typical atrial flutter with the goal of reducing the ventricular rate in the case of recurrence. The degree of modification of AV node conduction variables can be measured after the procedure and remains stable during the follow-up period. The failure rate of the procedure was 15% in the study patients. Prompt recognition of failure can guide the clinician's decision of whether to perform a His bundle ablation without resorting to an additional procedure. It should be emphasized that modulation of the AV node, because it does not prevent recurrence of atrial fibrillation, does not obviate the need for anticoagulation. Although the published data are promising, the long-term safety and effectiveness of the technique described herein require confirmation in a larger series with a longer follow-up period.

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